PHILADELPHIA NEUROLOGICAL SOCIETY.

October 22, 1901.

The President, Dr. James Tyson, in the chair.

Dr. John K. Mitchell showed a book from which one of his patients had cut out the figure three or multiples of three wherever he could find them.

Dr. F. X. Dercum stated that Dr. Mitchell's case was evidently one which was to be classified with the neurasthenic or neurasthenic-neuropathic insanities. Similar obsessions to those presented by Dr. Mitchell's patient are met with among the so-called "counters" and "mathematicians" who manifest irresistible enumeration, and have been described by Régis, Legrand du Saule and others.

Dr. Wm. Pickett read a study of paresis occurring in 149 cases at the Philadelphia Hospital.

Dr. Charles K. Mills said he did not think that Dr. Pickett had fully maintained by his statistics one or two of the points discussed. With regard to the demented type of general paresis, for instance, Dr. Mills was not entirely satisfied that the cases classed by Dr. Pickett as instances of general paresis were always true examples of this disease, as his crucial tests were not closely applied in making his differentiation. He gave for instance a little more than fifty per cent. of cases in which pupillary phenomena were present, but he failed to show how many of the cases constituting this percentage were included in his list of patients of the demented type, that is, cases without the classical delusions and usual course. Dr. Mills further said that it was well known that he (Dr. Mills) was a believer in the syphilitic origin of general paresis in perhaps eighty-five per cent. to ninety per cent. of the cases. He believed, however, that hospital statistics were of little value in determining this fact. His own conviction had come not only from a study of the literature of those who believed in the syphilitic origin of the disease, but especially from his experience of nearly a quarter of a century in private practice. It was much easier to get reliable statistics as to the existence of syphilis in the previous history of paretics in private practice than in hospital or dispensary work.

Dr. F. X. Dercum said he had occasionally employed the term "quiet cases of paresis" in his lectures, though in his published writings he had used the expression of "simple demented form" of paresis. All of the forms of paresis are demented, and when we speak of "simple demented form" we mean, of course, those cases in which the expansive or depressive delusions are absent. The number of the simple demented cases Dr. Dercum was convinced was quite large. He thought it was doubtful, however, whether it was as large as the statements of Dr. Pickett or Dr. Mendel would indicate. We should remember that many cases which are confined in the asylums have passed through expansive or depressive periods, short perhaps in duration, and of which periods there is no history or a very inadequate one. In studying paresis we should also bear in mind the occurrence of that interesting though rare form, circular paresis, of which Dr. Dercum had seen one example. In this patient cycle after cycle of depression and expansion followed in succession, until finally a simple dementia was established.

Dr. Dercum did not believe that there is any relationship between epilepsy and paresis. It is not surprising that paresis may occur in an epileptic, just as paresis may supervene in patients already suffering from other organic or visceral diseases. The fact that history of epilepsy followed by paresis is so rare is of sufficient significance.

He also believed that the term alcoholic paresis is intrinsically incorrect. There is not any direct causal relation between paresis and alcoholism. Paresis is a disease which bears definite relations to syphilis, not to alcoholism. Alcoholic dementia is one thing, paresis is another. Further, almost all, if not all, male paretics drink to excess during the earlier period of their disease. It is not surprising, therefore, that we should find occasionally elements present in the delusions which suggest alcoholism, such as the delusion of marital infidelity. However, the physical stigmata of paresis are unmistakable, and the rarity of alcoholic elements in the mental phenomena is again significant of the unimportant rôle played by alcohol.

In regard to idiopathic confusional insanity Dr. Dercum went further than to make the statement that it is rare. He believed that it does not exist.

In regard to heredity he may have been misunderstood by Dr. Pickett. He did not believe that paresis as paresis is hereditary. He did believe, however, that inherited neuropathies are of great importance in rendering the patient vulnerable to paresis.

Dr. Alfred Gordon thought that Dr. Pickett's paper would have been more complete if Dr. Pickett had added to his exhaustive study the question of the influence of hereditary syphilis in paresis. In the *Archives de Neurologie*, June, 1901, Régis published his fifth case of juvenile paresis occurring in a patient with a clear history of hereditary syphilis. The patient was a young man of twenty-three years, whose parents were distinctly syphilitic. Until twenty years of age he had been considered very intelligent, but at that period he became mentally incapacitated and gradually developed paresis. This case corroborates the general opinion that juvenile paresis takes generally a demented form.

In reference to pupillary changes Dr. Gordon wished to state that several years ago he made a comparative study of pupillary changes in all forms of insanity and paresis. The striking results oftained were those concerning paresis. While in other forms of insanity Dr. Gordon found very frequently pupillary inequalities there was rarely myosis or mydriasis. In paresis, on the contrary, pupillary inequalities were almost always associated with myosis or mydriasis of one or both eyes. Whether this observation can be of some aid for diagnostic purposes he did not know, but it deserves mention for future investigations.

A Case of Multiple Lesions of the Spinal Cord and Cranial Nerves, with Amyotrophy, Probably due to Syphilitic Infection.—Dr. Max. H. Bochroch and Dr. Alfred Gordon read a paper with the above title.

Supraorbital Reflex.—Dr. Joseph Sailer read a paper on the supraorbital reflex.

Tumor of the Frontal Lobe.—Drs. F. X. Dercum and W. W. Keen reported two cases of tumor of the frontal lobe.

Dr. Charles K. Mills said that it appeared that the osteoplastic flap in both of these cases had been made so as to uncover the motor region, the posterior line of the opening being at a position some distance back of the central fissure. It seemed to him that in both of these cases the posterior limits of the opening should have been the central fissure, as the localizing symptoms clearly pointed to the prefrontal region. Motor agraphia or orthographia was present in the former, with special psychical symptoms, and no persisting paralysis of any sort. The tumor was an enucleable one, and therefore the changing condition of motor agraphia was to be explained by the fact that the symptom was due more to pressure on, than to destruction of, the motor agraphic region. In the second case the symptoms were chiefly psychical. A tendency seemed to be exhibited to confine operations to uncovering the motor area if the study of the cases presented motor symptoms of any sort. We should by this time have reached a more advanced position in our efforts at localization. We can at least map out five distinct areas on the lateral convexity of the hemisphere for osteoplastic operations, namely: (1) an area whose posterior limit is the central fissure; (2) an area whose anterior limit is the central fissure or point just anterior to this fissure; (3) an area which uncovers the angular convolution; (4) an area which uncovers the occipital lobe; and (5) an area which uncovers the posterior extremities of the first and second temporal convolutions and adjoining region. In the absence of astereognosis and persisting motor symptoms the clinical phenomena present in both of these cases pointed unmistakably to the region in front of the central fissure. Why, therefore, trephine for one or two inches posterior to this fissure, necessitating in the end an unusually large opening, rongeuring, and therefore loss of time?